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The journey to psychosis: an exploration of specific psychological pathways

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Abstract Recent models of psychosis have implicated specific psychological processes in the aetiology of this disorder, and these factors may form a route to later symptoms—either directly or via a mediating pathway after exposure to adversity. Researchers are beginning to bring together findings that look into specific pathways between early experiences of adversity and different symptoms of psychosis, including thought disorder, hallucinations and persecutory delusions. The adversity-specific pathways include parental communication deviance, source monitoring biases, and insecure attachment. Researchers have also begun to utilise specific psychological factors as targets for treatment, and these include a focus on a worrying thinking style, negative beliefs about the self, interpersonal sensitivity, sleep disturbance, anomalous internal experience, and reasoning biases. Research on the impact of psychological processes is beginning to mount and is likely to improve our understanding of aetiology and lead to significant advances in the treatment of psychotic symptoms and disorders.

Keywords Psychosis · Adversity · Mechanisms · Thought disorder · Hallucinations · Delusions

Introduction

Cognitive models of psychosis have implicated specific psychological factors in the origins of this disorder [e.g. 1–5]. On the back of these theoretical propositions, there is now a mounting evidence base that is empirically evaluating the role of psychological processes in various pathways to psychosis—either through their direct impact on the development of specific psychotic symptoms, as discussed in the Freeman and Garety review [6], or via a more indirect route as potential mediators between adversity and psychosis, as addressed by the Bentall et al. review [7]. The issues presented in these two reviews are both important and timely. Due to increasing evidence in recent years of a connection between traumatic experiences and the development of psychosis, it is important to uncover specific factors that may increase risk for psychosis after exposure to trauma. While several literature reviews on the topic of trauma and psychosis do exist [e.g., 8, 9], there has yet to be a review which addresses the wider context by considering potential psychological mechanisms which may link adversity to psychosis. The Bentall et al. review [7] comprehensively fills this gap. Uncovering the mechanistic pathways between trauma and psychosis would accelerate translation by informing treatment priorities and future research [6].

An individual symptom approach

Both reviews stress the importance of studying the impact of potential aetiological mechanisms at the level of

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individual symptoms, rather than being diagnosis led. Psychosis is a heterogeneous phenomenon covering a wide range of psychiatric diagnoses, and therefore, its symptoms are unlikely to be a product of a single causal process, with different risk factors potentially increasing risk for different psychotic symptoms. Focusing on individual symptoms is a common approach adopted by psychologists when researching and treating mental health problems and has recently been strongly advocated by the US National Institute of Mental Health through their Research Domain Criteria strategy (<http://www.nimh.nih.gov/research-priorities/rdoc/index.shtml>). This strategic plan appeals for researchers to concentrate on dimensions of psychopathology and functioning that transcend traditional diagnostic boundaries. The aim of such approaches is to improve translation of research into effective treatment and the Freeman and Garety review [6] summarises tantalising preliminary evidence that this is indeed achievable (at least for persecutory delusions). A specific symptom approach is also important when considering the impact of different adversities as these varied exposures are unlikely to act on the same emotional and cognitive processes. Indeed, the evidence outlined in the Bentall et al. review [7] clearly underlines this point by presenting findings of differential mechanistic pathways to psychosis following exposure to different types of adverse experiences.

Specific risk pathways from adversity to psychosis

There has been consistent evidence in recent years which suggests an association between childhood trauma and psychosis [8]. However, it is crucial to understand whether there are differential effects of specific traumas on psychotic symptoms and elucidate the mechanisms underlying these associations, to advance our aetiological understanding of psychosis and learn how to improve current treatment options. For the majority of individuals, exposure to childhood adversity occurs many years before the development of psychosis, and this indicates that there must be other intervening factors which mediate or moderate this relationship. Bentall et al. [7] comprehensively summarise a range of potential mechanistic pathways between specific forms of childhood adversity and individual psychotic symptoms, including thought disorder, auditory verbal hallucinations (AVHs) and paranoia/persecutory delusions.

From the Bentall et al. review [7], it is clear that a solid research base is building when it comes to linking exposure to different adversities to different symptoms of psychosis, but unfortunately there have been very few studies to investigate the mediators that may exist between adversity and different manifestations of psychosis. There appears to

be most evidence for the role of dissociation as a potential mediator between childhood trauma and AVHs [10, 11], and also for current attachment style as a mediator between childhood neglect and later paranoia [12]. However, the evidence for a specific risk pathway between parental miscommunication and later thought disorder in offspring, and also for source monitoring biases as a mediator between childhood sexual abuse and AVHs, is reasonably sparse [7, 10]. It is evident that all of the proposed mechanisms within the Bentall et al. review [7] require further investigation, ideally by utilising longitudinal designs, as many have been proposed as potential pathways without any empirical support, or without consistent and robust evidence. However, all of the proposed candidates do make plausible sense as potential mechanisms, and are supported by theoretical models, and therefore, represent exciting avenues for future research.

An alternative pathway via adult adversity

Another pathway to psychosis that the Bentall et al. review [7] did not focus on is the potential mediating and/or moderating role of adult life events in the development of psychotic symptoms following exposure to childhood adversity. Exposure to significantly threatening life events in adulthood has long been known to be associated with an increased risk of psychosis, and a recent meta-analysis suggested around a threefold increase in the odds of psychosis and psychotic experiences after exposure to recent events [13]. Early and later exposure to adverse life events are likely to combine in complex ways to increase the risk of psychosis [14], and a recent study supports this suggestion by finding both a mediating and a synergistic role for adult adversity in the pathway between parental separation and later psychosis [15]. The association found between childhood and adult adversity, and psychosis, could arise as a result of cognitive and affective processes highlighted by contemporary psychological models of psychosis [3, 4]. It has been proposed that negative core schemas are formed early in life and may result from adverse experiences in childhood. If an individual experiences further trauma later in life, these schemas could become (re)activated, leading to emotional changes which may not only cause the development of psychotic experiences, but alter the appraisal of these anomalous occurrences, further increasing distress, and preventing a benign explanation from being concluded.

Research supports the possibility of negative schemas as a mediator of the relationship between early trauma and subclinical psychotic experiences [16–18], but no studies to date have tested the potential modifying effects of schemas after exposure to further victimisation in adulthood.

Research studies also support a mediational relationship between early adversity and psychosis via depression and anxiety [19, 20], but a pathway from later adverse experiences to clinical disorder, via affective processes, has yet to be explored. Improved understanding of these pathways is imperative because it may enable psychological interventions to be better targeted at high-risk individuals to potentially prevent the emergence of psychosis.

How can psychological processes be used as targets for treatment?

Existing research on tackling psychological processes linked to psychosis has focused not on adversity-specific mechanisms, but on more general psychological factors that may increase risk for specific psychotic symptoms, with the focus of the Freeman and Garety review [6] being on persecutory delusions and paranoia. Many of the potential treatment targets for persecutory delusions are currently undergoing testing, but are not yet supported by robust and consistent evidence. The first major randomised control trial for targeting worry in those with persecutory delusions has shown some promise [21], while brief reasoning training has led to reductions in paranoia and the jumping to conclusions bias [22]. Negative self-beliefs have also been consistently linked to persecutory delusions [23, 24], but no research has focused on treating negative beliefs in particular, and only a proxy for this process (self-esteem) has been used as a target for treatment [25]. Insomnia [26] and interpersonal sensitivity [27] have also been associated with increased paranoia and initial studies of CBT interventions have provided promising results [28, 29]. However, clearly there remains a need for further research utilising randomised controlled designs.

Broadening out to biopsychosocial models of psychosis?

It should be noted that an approach which focuses solely on psychological processes, such as those described in the Freeman and Garety review [6], is only testing one aspect of the potential aetiology of psychosis. Genetic and social factors are also likely to play a role and, therefore, findings concerning psychological mechanisms should be viewed within a broader biopsychosocial model, such as the traumagenic model [30, 31], and perhaps also considering a sociodevelopmental pathway to psychosis [14]. Stress-induced dysregulation of the HPA-axis may lead to neurochemical changes, such as increased dopamine receptor densities and dopamine release [32], which mirror dopaminergic abnormalities seen in individuals experiencing psychosis [33]. Furthermore, associations between

adversity and psychosis may also be influenced by an individual's genetic susceptibility, perhaps as a result of an underlying variation in their DNA sequence or due to epigenetic variation in gene expression [34]. However, the mechanisms involved in the aetiology of psychosis are not mutually exclusive and may be linked to one another via a causal chain, or interact to increase the risk of disorder. Thus, to progress this work and deliver valuable therapeutic advances, collaborations will be required across multiple disciplines.

Conclusions

To improve aetiological understanding of psychosis and inform priorities for future interventions, it is important to gain a deeper understanding of specific symptoms and mechanistic pathways and the ways in which they interact with one another. The findings of these reviews make a persuasive case for exploring these concepts at a symptom, rather than diagnostic level, and it is likely that these findings will provide a springboard for more exciting and novel research. It is clear that certain adversities show some specificity for different symptoms, but much more work needs to be done to explore the mediating factors which may underlie these relationships. Specific psychological factors are already showing some promise as targets for the treatment of paranoia and persecutory delusions, and further progress in psychological and pharmaceutical interventions is likely to come about through focusing on symptom-specific mechanisms.

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